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=> file uspatful

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FULL ESTIMATED COST	0.21	0.21

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FILE COVERS 1971 TO PATENT PUBLICATION DATE: 20 Oct 2005 (20051020/PD)
FILE LAST UPDATED: 20 Oct 2005 (20051020/ED)
HIGHEST GRANTED PATENT NUMBER: US6957446
HIGHEST APPLICATION PUBLICATION NUMBER: US2005235390

CA INDEXING IS CURRENT THROUGH 20 Oct 2005 (20051020/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 20 Oct 2005 (20051020/PD)
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USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Aug 2005

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>>> applications. USPAT2 contains full text of the latest US <<<
>>> publications, starting in 2001, for the inventions covered in <<<
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>>> classifications, or claims, that may potentially change from <<<
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This file contains CAS Registry Numbers for easy and accurate
substance identification.

```
=> s FC(w)gamma(w)RIIa
         50010 FC
         19577 FCS
         64972 FC
                  (FC OR FCS)
        228507 GAMMA
           838 GAMMAS
        228578 GAMMA
                  (GAMMA OR GAMMAS)
           252 RIIA
             1 RIIAS
           252 RIIA
                  (RIIA OR RIIAS)
L1         212 FC(W)GAMMA(W)RIIA

=> d kwic

L1   ANSWER 1 OF 212  USPATFULL on STN
SUMM    . . . FcγRIB and FcγRIC) are clustered in region 1q21.1
       of the long arm of chromosome 1; the genes encoding FcγRII
       isoforms (Fc.gamma.RIIA, FcγRIIB and
       FcγRIIC) and the two genes encoding FcγRIII (FcγRIIIA
       and FcγRIIIB) are all clustered in region 1q22. These different.
       . .
DRWD    FIG. 16A shows the binding of immune complexes using different
       antigen-antibody pairs to recombinant GST fusion protein of the
       Fc.gamma.RIIA receptor α subunit. FIG.
       16B shows the binding of the same antigen-antibody pairs to the GST
       fusion protein of the. . .
DRWD    . . . the different FcγRs. Binding of alanine variants at
       residues in the CH2 domain of anti-IgE E27 IgG1 are shown to Fc
       .gamma.RIIA, FcγRIIB, and FcγRIIIA. Type
       1 abrogates binding to all three receptors: D278A (265 in EU numbering).
       Type 2 improves binding to Fc.gamma.RIIA
       and FcγRIIB, while binding to FcγRIIIA is unaffected: S280A
       (267 in EU numbering). Type 3 improves binding to Fc.
       gamma.RIIA and FcγRIIB, but reduces binding to
       FcγRIIIA: H281A (268 in EU numbering). Type 4 reduces binding to
       Fc.gamma.RIIA and FcγRIIB, while
       improving binding to FcγRIIIA: S317A (298 in EU numbering). Type 5
```

improves binding to FcγRIIIA, but does not affect binding to **Fc.gamma.RIIA** and FcγRIIB: E352A, K353A (333 and 334 in EU numbering).

DETD . . . of the FcγRI, FcγRII, and FcγRIII subclasses, including allelic variants and alternatively spliced forms of these receptors. FcγRII receptors include **Fc.gamma.RIIA** (an "activating receptor") and FcγRIIB (an "inhibiting receptor"), which have similar amino acid sequences that differ primarily in the cytoplasmic domains thereof. Activating receptor **Fc.gamma.RIIA** contains an immunoreceptor tyrosine-based activation motif (ITAM) in its cytoplasmic domain. Inhibiting receptor FcγRIIB contains an immunoreceptor tyrosine-based inhibition motif. . .

DETD . . . affinity of the analyte for the receptor is relatively weak, e.g. in the micromolar range as is the case for **Fc.gamma.RIIA**, FcγRIIB, FcγRIIIA and FcγRIIIB. The method involves the formation of a molecular complex that has an improved avidity for the. . .

DETD This assay determines binding of an IgG Fc region to recombinant **Fc.gamma.RIIA**, FcγRIIB and FcγRIIIA α subunits expressed as His6-glutathione S transferase (GST)-tagged fusion proteins. Since the affinity of the Fc region. . . anti-IgG in a standard ELISA format (Example 2 below). The affinity of the other members of the FcγR family, i.e. **Fc.gamma.RIIA**, FcγRIIB and FcγRIIIA for IgG is however in the micromolar range and binding of monomeric IgG1 for these receptors can. . .

DETD . . . into larger molecular weight complexes via the chimeric IgE Fab:VEGF interaction. The E27 component of this complex binds to the **Fc.gamma.RIIA**, FcγRIIB and FcγRIIIA a subunits with higher avidity to permit detection in an ELISA format.

DETD . . . to C1q and hence did not activate complement were examined for their ability to bind to the Fc receptors: FcγRI, **Fc.gamma.RIIA**, FcγRIIB, FcγRIIIA and FcRn. This particular study was performed using a humanized anti-IgE antibody, an IgG1 antibody with these mutations. . .

DETD . . . the present study, the effect of mutating various Fc region residues of an IgG1 antibody with respect to binding FcγRI, **Fc.gamma.RIIA**, FcγRIIB and FcγRIIIIA as well as FcRn was evaluated. Antibody variants with improved as well as diminished FcR binding were. . .

DETD Low Affinity FcγR Binding ELISAs: **Fc.gamma.RIIA**, FcγRIIB and FcγRIIIA binding ELISAs were performed as described in Example 1 above, with detection of the stable hexamer (consisting. . .

DETD . . . additional variant, T307Q, also displayed improved FcRn binding compared to E27 parent antibody.

TABLE 6

CH2 DOMAIN VARIANTS

Res#EU **gamma.RIIA**		FcRn			FcγRI			**Fc.**	
IG2	(Kabat)	FcγRIIB mean	sd	n	mean	sd	n	mean	sd
		FcγRIIIA mean	sd						

REDUCED BINDING TO ALL. . .

DETD

TABLE 7

CH3 DOMAIN VARIANTS

Res#EU **gamma.RIIA**		FcRn			FcγRI			**Fc.**	
IG2	(Kabat)	FcγRIIB mean	sd	n	mean	sd	n	mean	sd
		FcγRIIIA mean	sd						

B1 K338(358)A 1.14 0.90. . .
DETD . . . and the FcR binding activity of those variants is summarized in the following table.
TABLE 8

NON-ALANINE VARIANTS

IG2	Res#EU (Kabat)	FcRn mean	sd	n	FcγRI mean	sd	n	**Fc.gamma.RIIA** FcγRIIB mean	sd	**Fc.gamma.RIIA** FcγRIIIA mean	sd
222	D249(262)E							0.97		0.99. . .	

DETD
TABLE 9

COMBINATION VARIANTS

IG2	Res#EU (Kabat)	FcRn mean	sd	n	FcγRI mean	sd	n	**Fc.gamma.RIIA** FcγRIIB mean	sd	**Fc.gamma.RIIA** FcγRIIIA mean	sd
96	S267(280)A							1.41		1.72. . .	

DETD This study includes a complete mapping of human IgG1 for human FcγRI, **Fc.gamma.RIIA**, FcγRIIB, FcγRIIIA, and FcRn. An alanine-scan of all amino acids in human IgG1 Fc (CH2 and CH3 domains) exposed to. . . FcγRI and FcRn are high affinity receptors and monomeric IgG could be evaluated in the assays for these two receptors. **Fc.gamma.RIIA**, FcγRIIB and FcγRIIIA are low affinity receptors and required use of an immune complex. Hence, an ELISA-type assay was used for **Fc.gamma.RIIA**, FcγRIIB, and FcγRIIIA, in which pre-formed hexamers, consisting of three anti-IgE E27 and three IgE molecules were bound to the. . .. al., Growth Factors 7:53 (1992) and Kim et al. Nature 362:841 (1993)). VEGF:anti-VEGF multimers also bound to the low affinity **Fc.gamma.RIIA** and FcγRIIIA (FIGS. 16A and 16B).
DETD . . . presence of specific allelic forms (reviewed in Lehrnbecher et al. Blood 94(12):4220-4232 (1999)). Several studies have investigated two forms of **Fc.gamma.RIIA**, R131 and H131, and their association with clinical outcomes (Hatta et al. Genes and Immunity 1:53-60 (1999); Yap et al.. . . et al. J. Clin. Invest. 100(5):1059-1070 (1997)). In this example, selected IgG variants were tested against both allelic forms of **Fc.gamma.RIIA** or FcγRIIIA. Fc receptor binding assays were performed essentially as described in the above examples. However, for FcγRIIIA-V158, both (a). . . FcγRIIIA-V158) were carried out. The results of these studies are summarized in Table 10 below.
TABLE 10

Binding of Variants to **Fc.gamma.RIIA** and FcγRIIIA Polymorphic Receptors

IG2	Res#EU (Kabat)	IgG Complex **Fc.gamma.RIIA**-H131 FcγRIIIA-V158 mean	sd	n	IgG Complex **Fc.gamma.RIIA**-R131 FcγRIIIA-V158 mean	sd	n	IgG Complex IgG Monomer FcγRIIIA-F158 mean	sd	n
11	T256(269)A. . .									

DETD . . . to the FcγRIIIA-F158 form also showed improved binding to the FcγRIIIA-V158 form though the improvement was not as

pronounced. For **Fc.gamma.RIIA**-R131 (used in assaying all variants) and **Fc.gamma.RIIA**-H131, the binding pattern of the selected IgG1 variants did show some distinct differences. S267(280)A, H268(281)A, and S267(280)A/H268(281)A exhibited improved binding to **Fc.gamma.RIIA**-R131, compared to native IgG1, but not to **Fc.gamma.RIIA**-H131. In contrast, S267(280)G showed improved binding to **Fc.gamma.RIIA**-R131 but reduced binding to **Fc.gamma.RIIA**-H131 (Table 10). Other variants bound similarly to both allelic **Fc.gamma.RIIA** forms: V305(324)A, T307(326)A, N315(324)A, K317(336)A, and K320(339)A.

```
=> s FC(w)gamma(w)RIIa (7a) receptor
         50010 FC
         19577 FCS
         64972 FC
                 (FC OR FCS)
        228507 GAMMA
           838 GAMMAS
        228578 GAMMA
                 (GAMMA OR GAMMAS)
           252 RIIA
             1 RIIAS
           252 RIIA
                 (RIIA OR RIIAS)
        119017 RECEPTOR
         86804 RECEPTORS
        135368 RECEPTOR
                 (RECEPTOR OR RECEPTORS)
L2         167 FC(W)GAMMA(W)RIIA (7A) RECEPTOR

=> s l2/clm and (inhibitor or antagonist or composition)/clm
          4476 FC/CLM
           187 FCS/CLM
          4656 FC/CLM
                 ((FC OR FCS)/CLM)
         30568 GAMMA/CLM
            36 GAMMAS/CLM
         30577 GAMMA/CLM
                 ((GAMMA OR GAMMAS)/CLM)
            37 RIIA/CLM
         25401 RECEPTOR/CLM
          6442 RECEPTORS/CLM
         28510 RECEPTOR/CLM
                 ((RECEPTOR OR RECEPTORS)/CLM)
            17 FC/CLM(W)GAMMA/CLM(W)RIIA/CLM (7A) RECEPTOR/CLM
         24666 INHIBITOR/CLM
          7871 INHIBITORS/CLM
         29823 INHIBITOR/CLM
                 ((INHIBITOR OR INHIBITORS)/CLM)
          8781 ANTAGONIST/CLM
          2570 ANTAGONISTS/CLM
         10416 ANTAGONIST/CLM
                 ((ANTAGONIST OR ANTAGONISTS)/CLM)
        338169 COMPOSITION/CLM
         20320 COMPOSITIONS/CLM
        343333 COMPOSITION/CLM
                 ((COMPOSITION OR COMPOSITIONS)/CLM)
L3          12 (FC/CLM(W)GAMMA/CLM(W)RIIA/CLM (7A) RECEPTOR/CLM) AND (INHIBITOR
               OR ANTAGONIST OR COMPOSITION)/CLM

=> d bib,kwic 1-12

L3   ANSWER 1 OF 12  USPATFULL on STN
AN     2005:204449  USPATFULL
TI     Fcyriia transgenic animal model for autoimmune disease
```

IN Hogarth, Phillip Mark, Victoria, AUSTRALIA
Mottram, Patricia Lesley, Victoria, AUSTRALIA
Sardjono, Caroline Tan, West Java, INDONESIA
PI US 2005177876 A1 20050811
AI US 2003-517251 A1 20030606 (10)
WO 2003-AU718 20030606
PRAI AU 2003-2856 20020607
AU 2003-2002950529 20020801
DT Utility
FS APPLICATION
LREP SHERIDAN ROSS PC, 1560 BROADWAY, SUITE 1200, DENVER, CO, 80202, US
CLMN Number of Claims: 42
ECL Exemplary Claim: 1
DRWN 14 Drawing Page(s)
LN.CNT 1445
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:

. . . of: a) administering a compound to be screened to a non-human transgenic animal that has been modified to express human **Fc**. **gamma**.**RIIa receptor** such that the transgenic animal is susceptible to an autoimmune disease; and b) assessing the transgenic animal to determine if. . .

. . . of: a) administering a compound to be screened to a non-human transgenic animal that has been modified to express human **Fc**. **gamma**.**RIIa receptor** such that the transgenic animal is susceptible to an autoimmune disease; and b) assessing the transgenic animal to determine if. . .

. . . disease, the method comprising the steps of: a) administering a compound to be screened to a non-human cell expressing human **Fc** .**gamma**.**RIIa receptor**, wherein the cell is derived from a non-human transgenic animal that has been modified to express human **Fc**.**gamma**.**RIIa receptor** such that the transgenic animal is susceptible to an autoimmune disease; and b) assessing the cell to determine if the. .

. . . claim 1, wherein the non-human transgenic animal is resistant to collagen-induced arthritis prior to being modified to express the human **Fc**.**gamma**.**RIIa receptor**.

. . . transgenic animal is a transgenic mouse derived from the strains C57BL/6 and SJL that has been modified to express human **Fc**. **gamma**.**RIIa receptor**.

21. A **composition** for treating or preventing an autoimmune disease, the **composition** comprising an effective amount of a compound that can reduce aberrant immune activity in an animal, and a pharmaceutically acceptable. . .

22. A **composition** according to claim 21, wherein the compound can reduce aberrant immune complex formation, aberrant immune complex clearance or immune complex. . .

23. A **composition** according to claim 21, wherein the compound can reduce aberrant immune activity in the animal by inhibiting the activity of. . .

24. A **composition** according to claim 21, wherein the autoimmune disease is caused by aberrant immune complex formation, aberrant immune complex clearance or. . .

25. A **composition** according to claim 21, wherein the autoimmune disease is selected from the group consisting of arthritis and systemic lupus erythematosus. . .

26. A **composition** according to claim 21, wherein the autoimmune disease is rheumatoid arthritis (RA).

27. A **composition** according to claim 21, wherein the autoimmune disease is collagen-induced arthritis (CIA).

28. A non-human transgenic animal that has been modified to express human **Fc**.**gamma**.**RIIa receptor** such that the transgenic animal is susceptible to an autoimmune disease,

wherein the transgenic animal is resistant to collagen-induced arthritis prior to being modified to express the human **Fc.gamma .RIIa receptor**.

. . . 29, wherein the transgenic mouse is derived from the strains C57BL/6 and SJL that has been modified to express human **Fc. gamma.RIIa receptor**.

. . . transgenic animal model for autoimmune disease, the method comprising the steps of: a) introducing a nucleic acid molecule encoding human **Fc.gamma.RIIa receptor** to a cell of a non-human embryo; b) transferring the embryo to a foster mother; and c) assessing the resultant. . . non-human transgenic embryo is resistant to collagen-induced arthritis prior to the introduction of a nucleic acid molecule encoding a human **Fc.gamma. RIIa receptor**.

. . . transgenic animal is a transgenic mouse derived from the strains C57BL/6 and SJL that has been modified to express human **Fc. gamma.RIIa receptor**.

42. A method for producing a **composition** for treating or preventing an autoimmune disease, the method comprising: a) selecting the compound by the method according to claim 1; and b) formulating the compound with a pharmaceutically acceptable diluent, excipient or carrier to produce the **composition**.

L3 ANSWER 2 OF 12 USPATFULL on STN
AN 2005:195779 USPATFULL
TI Pecam-1 modulation
IN Gibbins, Jonathan M, School of Animal and Microbial Sciences,, The University of Reading, Whiteknights House, Reading, UNITED KINGDOM RG6 6AJ
Cicmil, Milenko, Reading, UNITED KINGDOM
PI US 2005169920 A1 20050804
AI US 2003-500027 A1 20021217 (10)
WO 2002-GB5730 20021217
PRAI GB 2003-130832 20011222
DT Utility
FS APPLICATION
LREP WORKMAN NYDEGGER, (F/K/A WORKMAN NYDEGGER & SEELEY), 60 EAST SOUTH TEMPLE, 1000 EAGLE GATE TOWER, SALT LAKE CITY, UT, 84111, US
CLMN Number of Claims: 19
ECL Exemplary Claim: 1-18
DRWN 7 Drawing Page(s)
LN.CNT 664
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
. . . wherein the activator is selected from the group comprising; a small molecule, an antibody, an antibody derivative, an agonist, an **antagonist**, a ligand, a DNA sequence, a complementary DNA sequence, an antisense DNA sequence, a probe, a protein sequence, a recombinant. . . PECAM-1, a catalyst, shear, oxidative stress, FcεRI, the high affinity receptor for FcεRI, an activated form of the high affinity **receptor Fc. gamma.RIIA**, **Fc.gamma.RIIA** , the low affinity **receptor** for **Fc.gamma. RIIA** and an activated form of the low affinity **receptor Fc.gamma.RIIA**.

L3 ANSWER 3 OF 12 USPATFULL on STN
AN 2005:63790 USPATFULL
TI Optimized Fc variants and methods for their generation
IN Lazar, Gregory Alan, Glendale, CA, UNITED STATES
Chirino, Arthur J., Camarillo, CA, UNITED STATES
Dang, Wei, Pasadena, CA, UNITED STATES

Desjarlais, John R., Pasadena, CA, UNITED STATES
Doberstein, Stephen Kohl, Pasadena, CA, UNITED STATES
Hayes, Robert J., Radnor, PA, UNITED STATES
Karki, Sher Bahadur, Pasadena, CA, UNITED STATES
Vafa, Omid, Monrovia, CA, UNITED STATES
PA Xencor, Inc. (U.S. corporation)
PI US 2005054832 A1 20050310
AI US 2004-822231 A1 20040326 (10)
RLI Continuation-in-part of Ser. No. US 2003-672280, filed on 26 Sep 2003, PENDING Continuation-in-part of Ser. No. US 2003-379392, filed on 3 Mar 2003, PENDING
PRAI US 2003-477839P 20030612 (60)
US 2003-467606P 20030502 (60)
US 2002-414433P 20020927 (60)
US 2003-442301P 20030123 (60)
DT Utility
FS APPLICATION
LREP Robin M. Silva, Esq., Dorsey & Whitney LLP, Intellectual Property Department, Four Embarcadero Center, Suite 3400, San Francisco, CA, 94111-4187
CLMN Number of Claims: 37
ECL Exemplary Claim: 1
DRWN 32 Drawing Page(s)
LN.CNT 8390
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
. . . wherein said Fc variant binds with greater affinity to human FcγRI and FcγRIIIa, but exhibits unaltered affinity to a human **receptor** selected from the group consisting of **Fc.gamma.RIIa**, FcγRIIb, and FcγRIIc.

11. A variant protein according to claim 3, wherein said Fc variant binds with greater affinity to human **Fc.gamma.** **RIIa**, but exhibits unaltered affinity to a human **receptor** selected from the group consisting of FcγRI, FcγRIIb, FcγRIIc, and FγRIIa.

36. A pharmaceutical **composition** comprising a variant protein according to claim 1 and a pharmaceutically acceptable carrier.

L3 ANSWER 4 OF 12 USPATFULL on STN
AN 2004:202937 USPATFULL
TI Treatment of patients with multiple sclerosis based on gene expression changes in central nervous system tissues
IN Dangond, Fernando, Newton, MA, UNITED STATES
Hwang, Daehee, Seattle, WA, UNITED STATES
Gullans, Steven R., Natick, MA, UNITED STATES
PI US 2004156826 A1 20040812
AI US 2003-670766 A1 20030925 (10)
PRAI US 2002-414219P 20020927 (60)
DT Utility
FS APPLICATION
LREP FULBRIGHT & JAWORSKI L.L.P., SUITE 2400, 600 CONGRESS AVENUE, AUSTIN, TX, 78701-3271
CLMN Number of Claims: 56
ECL Exemplary Claim: 1
DRWN 2 Drawing Page(s)
LN.CNT 7243
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
1. A method for treating or preventing multiple sclerosis (MS) comprising administering to a subject with MS a **composition** that causes an increase in the activity or expression of at least one gene product selected from the group consisting. . .
3. The method of claim 2, wherein the second MS therapy is given before the **composition**.

4. The method of claim 2, wherein the second MS therapy is given after the **composition**.

5. The method of claim 2, wherein the second MS therapy is given concurrent with the **composition**.

7. The method of claim 1, wherein the **composition** comprises peptide.

8. The method of claim 1, wherein the **composition** comprises a small molecule.

9. The method of claim 1, wherein the **composition** is an organo-pharmaceutical.

10. The method of claim 1, wherein the **composition** comprises an expression cassette comprising a nucleic acid encoding the selected gene product and a promoter active in eukaryotic cells,. . .
17. The method of claim 1, wherein said **composition** is administered intradermally, subcutaneously, intramuscularly, intraperitoneally, intravenously, intranasally, intraalveolarly, parenterally, intrathecally, intraparenchymally or intraperitoneally.

18. The method of claim 1, wherein said **composition** is administered to said mammal more than once.

19. The method of claim 1, wherein said **composition** is administered to said mammal in discrete repeated dosings.

. . claim 1 or 20, wherein at least one gene product comprises cyclin E (X95406), thymocyte antigen CD1a (M28825), serine protease **inhibitor** p19 (U71364), and skeletal muscle troponin T (M21984).

. . protein (NeuroD2) (U58681), H-neuro-d4 (U43843), Lim-Domain Transcription Factor Lim-1 (HG4318-HT4588), Cyclin D3 (M92287), Cyclin E1 (M74093), Cyclin G1 (X77794), Kinase **Inhibitor** P27kip1 Cyclin-Dependent (HG4258-HT4528), Cdk-**inhibitor** p57KIP2 (KIP2) (U22398), HsMcm6 (D84557), Retinoblastoma related protein (p107) (L14812), thymidylate synthase-inducer transcription factor LSF (U03494), 218 kD Mi-2 protein. . . heparan sulfate proteoglycan (X62515), Guanylate kinase (GUK1) (L76200), RAD23A homolog (AD000092), Mismatch repair protein (hMLH1) (AF001359), thymidylate kinase (CDC8) (L16991), **Inhibitor** of apoptosis protein 1 (U45878), Lysosome-associated membrane protein-2 (S79873), GM2 activator protein (X62078), alpha mannosidase (U37248), Alpha mannosidase II isozyme. .
.
25. A method for treating or preventing multiple sclerosis (MS) comprising administering to a subject with MS a **composition** that causes a decrease in the activity level or expression of a gene product selected from the group consisting of. . .
27. The method of claim 26, wherein the second MS therapy is given before the **composition**.

28. The method of claim 26, wherein the second MS therapy is given after the **composition**.

29. The method of claim 26, wherein the second MS therapy is given concurrent with the **composition**.

31. The method of claim 25, wherein the **composition** comprises peptide.

32. The method of claim 25, wherein the **composition** comprises a small molecule.

33. The method of claim 25, wherein the **composition** is an organo-pharmaceutical.

34. The method of claim 25, wherein the **composition** comprises an expression cassette comprising a nucleic acid encoding an antisense construct or a ribozyme targeting the selected gene product,. . .
41. The method of claim 25, wherein said **composition** is administered intradermally, subcutaneously, intramuscularly, intraperitoneally, intravenously, intranasally, intraalveolarly, parenterally, intrathecally, intraparenchymally or intraperitoneally.

42. The method of claim 25, wherein said **composition** is administered to said mammal more than once.

43. The method of claim 25, wherein said **composition** is administered to said mammal in repeated discrete dosings.

. . . light chain protein 14.1 (Ig lambda chain related) (M34516), IGHA1 from Ig germline H-chain G-E-A region A: gamma-3 5 (J00220), **Fc -gamma-RIIA** IgG Fc **receptor** class IIA (X68090), Fc **Receptor** Iib3 For Igg, Low Affinity (HG491-HT491), Ig-like transcript 2 (U82279), Ig Heavy Chain Vdjc Regions (HG4458-HT4727), Ig J chain (M12759),. . . . . HSP70B' (X51757), Thromboxane synthase (M80647), Thromboxane A2 receptor (D38081), Thrombospondin 2 (HG896-HT896), Granulocyte colony-stimulating factor receptor (CSF3R) (M59820), Plasminogen activator **inhibitor** type 1 N-terminus (X04729), Autoimmune Antigen Thyroid Disease-Related Antigen (HG3578-HT3781), Integrin beta-5 subunit (X53002), Integrin beta 7 subunit (S80335), Neuronal PAS1 (NPAS1) (U77968), Prointerleukin 1 beta (X04500), Interleukin 1 receptor **antagonist** IRAP (X53296), R kappa B (U08191), Cathepsin C (X87212), Lymphocyte Antigen Hla-G3 (HG273-HT273), Lymph node homing receptor (M25280), Monocyte chemoattractant protein-4 precursor (MCP-4) (U46767), thymosin beta (D82345), Tissue **inhibitor** of metalloproteinase 4 (U76456), Pancreatic phospholipase A-2 (PLA-2) (M21056), Fetal brain adenylyl cyclase (L05500), Adenylyl cyclase (L21993), Guanine nucleotide-binding protein. . . (M36429), Transducin-like enhancer protein (TLE3) (M99438), Low-Mr GTP-binding protein (RAB31) (U59877), 43 kDa inositol polyphosphate 5-phosphatase (Z31695), RAB7 (X93499), Ras **Inhibitor** Inf (HG511-HT511), R-ras (M14949), RasGTPase activating protein (D78156), Clone 110298 (L43579), Rab GDI (D13988), RhoE=26 kda GTPase homolog (S82240), HSPDE4C1. . . (M37238), GTPase activating protein (rap1GAP) (M64788), RasGTPase activating protein (D78156), Ras-Specific Guanine Nucleotide-Releasing Factor (HG2510-HT2606), Ras-related protein Rab5b (X54871), Ras **inhibitor** (Rin1) (L36463), RIN protein (Y07565), Guanine Nucleotide-Binding protein Rap2 (HG1996-HT2044), Guanine nucleotide-binding protein (Gi) alpha subunit (M27543), Guanine nucleotide-binding protein. . . protein (X90872), E6-AP ubiquitin protein ligase 3A (AF002224), Ribosomal DNA repeating unit (U13369), Vacuolar proton ATPase subunit D (X71490), Inter-alpha-trypsin **inhibitor** subunit 3 (X16260), Leukemia virus receptor 1 (GLVR1) (L20859), Clone S171 (L40393), Clone cD24-1 Huntington's candidate region fragment (L37199), FLII. . . 1 (X68149), Trabecular meshwork-induced glucocorticoid response protein (AF001620), bHLH-PAS protein Jap3 (U60415), Guanylin (M97496), Dioxin-responsive (S81578), RD/X99296 (X99296), Plasma inter-alpha-trypsin **inhibitor** heavy chain H(3) (X14690), Major Yo paraneoplastic antigen (CDR2) (M63256), PCI (plasminogen activator **inhibitor** 3) from protein C **inhibitor** (M68516), KNP-Ib; Also: U53003 (D86062), MJD1=MJD1 protein {CAG repeats} (S75313), POM121-like 1 (D87002), Cell surface glycoprotein P3.58 (M55024), Oviductal glycoprotein. . . Kallmann syndrome (KAL) (M97252), PACAP receptor (D17516), Retinal pigment epithelium-specific 61 kDa protein (RPE65) (U18991), Squamous cell carcinoma antigen=serine protease **inhibitor** (S66896), Clone 23948 sequence (U79293), Albumin, 3; Also: HG2841-HT2970, HG2841-HT2968 (HG2841-HT2969), Protein containing SH3 domain SH3GL2 (X99657), HK2 hexokinase II. . .
50. A method of identifying a **composition** useful in the treatment or prevention of multiple sclerosis (MS) comprising: (a)

providing a cell that expresses one or more. . . or more genes, wherein modulation of the expression of the one or more genes identifies said candidate substances as a **composition** useful in the treatment or prevention of multiple sclerosis MS.

L3 ANSWER 5 OF 12 USPATFULL on STN
AN 2004:64262 USPATFULL
TI Methods of inhibiting phagocytosis
IN Schreiber, Alan D., Philadelphia, PA, UNITED STATES
Park, Jong-Gu, Drexel Hill, PA, UNITED STATES
PA THE UNIVERSITY OF PENNSYLVANIA, Philadelphia, PA (U.S. corporation)
PI US 2004048781 A1 20040311
AI US 2003-639662 A1 20030813 (10)
RLI Continuation of Ser. No. US 2001-811492, filed on 20 Mar 2001, GRANTED, Pat. No. US 6638764 Continuation of Ser. No. US 1998-158980, filed on 14 Sep 1998, GRANTED, Pat. No. US 6242427 Continuation of Ser. No. US 1996-657884, filed on 7 Jun 1996, GRANTED, Pat. No. US 5858981 Continuation-in-part of Ser. No. US 1995-483530, filed on 7 Jun 1995, ABANDONED Continuation-in-part of Ser. No. US 1994-316425, filed on 30 Sep 1994, ABANDONED Continuation-in-part of Ser. No. US 1993-129381, filed on 30 Sep 1993, ABANDONED
DT Utility
FS APPLICATION
LREP NIXON & VANDERHYE, PC, 1100 N GLEBE ROAD, 8TH FLOOR, ARLINGTON, VA, 22201-4714
CLMN Number of Claims: 59
ECL Exemplary Claim: 1
DRWN 11 Drawing Page(s)
LN.CNT 1469
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
. . . in a mammal comprising introducing into phagocytic cells of said mammal that are in contact with said immune complexes an **inhibitor** of a kinase endogenous to said cells associated with an Fc receptor present at the membrane of said cells, said. . .
3. The method according to claim 1 wherein said **inhibitor** is a peptide or mimetic.

20. The method according to claim 19 wherein sa d Fc **receptor** is FcγRI, **Fc.gamma.RIIA** or FcγRIIIA.

22. The method according to claim 21 wherein said Fc **receptor** is FcγRI, **Fc.gamma.RIIA** or FcγRIIIA.

. . . the signal transduction of the γ subunit of the IgE receptor FcεRI comprising introducing into cells bearing said receptor an **inhibitor** of a kinase endogenous to said cells that activates said signal transduction of said FcεRI receptor or the γ subunit. . .
31. The method according to claim 30 wherein said **inhibitor** is a peptide or mimetic.

51. The method according to claim 47 wherein said **inhibitor** targets the interval region between the second SH2 domain and catalytic (kinase) domain of Syk kinase.

59. A pharmaceutical **composition** comprising Syk kinase interval region, or portion thereof of at least 6 amino acids, or mimetic thereof, and a pharmaceutically. . .

L3 ANSWER 6 OF 12 USPATFULL on STN
AN 2003:112552 USPATFULL
TI Use of bispecific antibodies to regulate immune responses
IN Bigler, Michael Eric, Redwood City, CA, UNITED STATES
Cherwinski, Holly Marie, Boulder Creek, CA, UNITED STATES

Phillips, Joseph H., Palo Alto, CA, UNITED STATES
PI US 2003077282 A1 20030424
AI US 2002-270084 A1 20021011 (10)
PRAI US 2001-329182P 20011012 (60)
DT Utility
FS APPLICATION
LREP DNAX RESEARCH, INC., LEGAL DEPARTMENT, 901 CALIFORNIA AVENUE, PALO ALTO, CA, 94304
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1432
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
5. The method of claim 1, wherein the activating receptor is selected from the group consisting of FcεRI, FcγRIII, **Fc**.
gamma.**RIIA**, FcγRIIC, T-cell **receptor**,
TREM-1, TREM-2, CD28, CD3, CD2, and DAP-12.

15. A **composition** comprising the bispecific antibody of claim 1 in conjunction with an acceptable carrier.

L3 ANSWER 7 OF 12 USPATFULL on STN
AN 2003:105832 USPATFULL
TI Genetic manipulation of phagocytes for modulation of antigen processing and the immune response therefrom
IN Albert, Matthew, New York, NY, UNITED STATES
Birge, Raymond, New York, NY, UNITED STATES
PI US 2003072743 A1 20030417
AI US 2002-238213 A1 20020910 (10)
RLI Continuation of Ser. No. US 2000-565958, filed on 5 May 2000, ABANDONED
DT Utility
FS APPLICATION
LREP KLAUBER & JACKSON, 411 HACKENSACK AVENUE, HACKENSACK, NJ, 07601
CLMN Number of Claims: 143
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3105
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
17. The method of claim 16 wherein said signaling domain derived from a member of the Fc **receptor** family is the FcγRI,
Fc.**gamma**.**RIIA**, FcγRIIB, or
FcγRIII α-chain.

34. The method of claim 33 wherein said signaling domain derived from a member of the Fc **receptor** family is FcRγI, **Fc**.
gamma.**RIIA**, FcγRIIB, or FcRγIII
α-chain.

53. The method of claim 52 wherein said signaling domain derived from a member of the Fc **receptor** family is FcRγI, **Fc**.
gamma.**RIIA**, FcγRIIB, or FcRγIII
α-chain.

. . . cells or precursors thereof to apoptotic cells expressing said antigen in the presence of at least one of the following **compositions**: i) an agent capable of both facilitating cross-priming and maturing said dendritic cell; or ii) the combination of at least. . .
135. The method of claim 134 wherein said signaling domain derived from a member of the Fc **receptor** family is the FcγRI,
Fc.**gamma**.**RIIA**, FcγRIIB, or
FcγRIII α-chain.

139. The integrin receptor heterodimer of claim 138 wherein said signaling domain derived from a member of the Fe **receptor**

family is the FcγRI, **Fc.gamma.RIIA**, FcγRIIB, or FcγRIII α-chain.

. . . The integrin receptor chimeric β subunit of claim 141 wherein said signaling domain derived from a member of the Fc **receptor** family is the FcγRI, **Fc.gamma.RIIA**, FcγRIIB, or FcγRIII α-chain.

L3 ANSWER 8 OF 12 USPATFULL on STN
AN 2002:235026 USPATFULL
TI Tripeptide of FcgammaRIIA
IN Schreiber, Alan D., Philadelphia, PA, UNITED STATES
Worth, Randall, Philadelphia, PA, UNITED STATES
Petty, Howard R., Detroit, MI, UNITED STATES
PI US 2002127209 A1 20020912
AI US 2001-989298 A1 20011121 (9)
PRAI US 2000-252460P 20001122 (60)
DT Utility
FS APPLICATION
LREP NIXON & VANDERHYE P.C., 1100 North Glebe Road, 8th Floor, Arlington, VA, 22201
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN 5 Drawing Page(s)
LN.CNT 614
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
4. The method according to claim 3 wherein said Fc **receptor** is **Fc.gamma.RIIA** or a modified form thereof comprising at least 1 additional L-T-L peptide in the cytoplasmic domain thereof.

13. The method according to claim 11 wherein said agent is IFN-γ or an **inhibitor** of IL-4.

L3 ANSWER 9 OF 12 USPATFULL on STN
AN 2002:133845 USPATFULL
TI Methods of inhibiting phagocytosis
IN Schreiber, Alan D., Philadelphia, PA, UNITED STATES
Park, Jong-Gu, Drexel Hill, PA, UNITED STATES
PA UNIVERSITY OF PENNSYLVANIA. (U.S. corporation)
PI US 2002068703 A1 20020606
US 6638764 B2 20031028
AI US 2001-811492 A1 20010320 (9)
RLI Continuation of Ser. No. US 1998-158980, filed on 14 Sep 1998, GRANTED, Pat. No. US 6242427 Continuation-in-part of Ser. No. US 1996-657884, filed on 7 Jun 1996, GRANTED, Pat. No. US 5858981 Continuation-in-part of Ser. No. US 1995-483530, filed on 7 Jun 1995, ABANDONED Continuation-in-part of Ser. No. US 1994-316425, filed on 30 Sep 1994, ABANDONED Continuation-in-part of Ser. No. US 1993-129381, filed on 30 Sep 1993, ABANDONED
DT Utility
FS APPLICATION
LREP Nixon & Vanderhye P.C., 8th Floor, 1100 N. Glebe Rd., Arlington, VA, 22201
CLMN Number of Claims: 59
ECL Exemplary Claim: 1
DRWN 11 Drawing Page(s)
LN.CNT 1477
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
. . . in a mammal comprising introducing into phagocytic cells of said mammal that are in contact with said immune complexes an **inhibitor** of a kinase endogenous to said cells associated with an Fc receptor present at the membrane of said cells, said. . .
3. The method according to claim 1 wherein said **inhibitor** is a

peptide or mimetic.

20. The method according to claim 19 wherein said Fc **receptor** is FcγRI, **Fc.gamma.RIIA** or FcγRIIIA.

22. The method according to claim 21 wherein said Fc **receptor** is FcγRI, **Fc.gamma.RIIA** or FcγRIIIA.

. . . the signal transduction of the γ subunit of the IgE receptor FcεRI comprising introducing into cells bearing said receptor an **inhibitor** of a kinase endogenous to said cells that activates said signal transduction of said FcεRI receptor or the γ subunit. . .
31. The method according to claim 30 wherein said **inhibitor** is a peptide or mimetic.

51. The method according to claim 47 wherein said **inhibitor** targets the interval region between the second SH2 domain and catalytic (kinase) domain of Syk kinase.

59. A pharmaceutical **composition** comprising Syk kinase interval region, or portion thereof of at least 6 amino acids, or mimetic thereof, and a pharmaceutically. . .

L3 ANSWER 10 OF 12 USPATFULL on STN
AN 2002:119856 USPATFULL
TI Fc receptor modulators and uses thereof
IN Baell, Jonathan B., Ivanhoe, AUSTRALIA
Garrett, Thomas P.J., Brunswick, AUSTRALIA
Hogarth, P. Mark, Williamstown, AUSTRALIA
Matthews, Barry R., Olinda, AUSTRALIA
McCarthy, Thomas D., East Malvern, AUSTRALIA
Pietersz, Geoffrey A., Greensborough, AUSTRALIA
PA Ilexus Pty Limited (non-U.S. corporation)
PI US 2002061844 A1 20020523
US 6835753 B2 20041228
AI US 2001-995277 A1 20011126 (9)
RLI Continuation of Ser. No. US 1999-393598, filed on 10 Sep 1999, PENDING
PRAI US 1998-99855P 19980911 (60)
US 1999-131938P 19990430 (60)
US 1999-148479P 19990811 (60)
DT Utility
FS APPLICATION
LREP SHERIDAN ROSS PC, 1560 BROADWAY, SUITE 1200, DENVER, CO, 80202
CLMN Number of Claims: 107
ECL Exemplary Claim: 1
DRWN 20 Drawing Page(s)
LN.CNT 2100
CLM What is claimed is:
1. A pharmaceutical **composition** comprising: (a) compound selected from the group consisting of an aromatic compound of the formula: ##STR50## a heteroaromatic compound of. . .
2. The **composition** of claim 1, wherein said compound is of the formula: ##STR55##

3. The **composition** of claim 2, wherein said compound is of the formula: ##STR56##

4. The **composition** of claim 3, wherein m and n are 0.

5. The **composition** of claim 4, wherein W.sup.1 and W.sup.2 are CO.sub.2H.

6. The **composition** of claim 5, wherein R.sup.1 and R.sup.2 are a bond.

7. The **composition** of claim 6, wherein L.sup.1 is --CH.sub.2CH.sub.2--.

8. The **composition** of claim 6, wherein L.sup.1 is --CH.sub.2O--.

9. The **composition** of claim 6, wherein L.sup.1 is --CH.dbd.CHC(.dbd.O)--.

10. The **composition** of claim 6, wherein L.sup.1 is --CH.sub.2CH.sub.2CH(OH)--.

11. The **composition** of claim 6, wherein L.sup.1 is --CH.dbd.CH--.

12. The **composition** of claim 6, wherein L.sup.1 is --CH(OH)CH(OH)--.

13. The **composition** of claim 12, wherein the stereochemistry of hydroxy groups is (S,S).

14. The **composition** of claim 6, wherein L.sup.1 is --CH.sub.2N(R.sup.26)CH.sub.2, wherein R.sup.26 is H, C.sub.1-C.sub.6alkyl or an amine protecting group.

15. The **composition** of claim 14, wherein R.sup.26 is --CH.sub.2CO.sub.2H.

16. The **composition** of claim 6, wherein L.sup.1 is a moiety of the formula: ##STR57##

17. The **composition** of claim 5, wherein R.sup.1 and R.sup.2 are --CH.sub.2--.

18. The **composition** of claim 17, wherein L.sup.1 is ethylene.

19. The **composition** of claim 17, wherein L.sup.1 is --CH.dbd.CH--.

20. The **composition** of claim 5, wherein R.sup.1 is methylene, R.sup.2 is a bond and L.sup.1 is ethylene.

21. The **composition** of claim 4, wherein W.sup.1 and W.sup.2 are PO(OR.sup.15).sub.2, and R.sup.1 and R.sup.2 are a bond.

22. The **composition** of claim 21, wherein L.sup.1 is ethylene.

23. The **composition** of claim 22, wherein R.sup.15 is ethyl.

24. The **composition** of claim 22, wherein R.sup.15 is H.

25. The **composition** of claim 21, wherein L.sup.1 is a moiety of the formula: ##STR58## wherein each of R.sup.27 and R.sup.28 is independently. . .

26. The **composition** of claim 25, wherein each of R.sup.27 and R.sup.28 is independently 4-methoxybenzyl or H.

27. The **composition** of claim 6, wherein L.sup.1 is a moiety of the formula: ##STR59## wherein each of R.sup.27 and R.sup.28 is independently. . .

28. The **composition** of claim 27, wherein each of R.sup.27 and R.sup.28 is independently 4-methoxybenzyl or H.

29. The **composition** of claim 4, wherein L.sup.1 is --CH.dbd.CH--, W.sub.1 and W.sub.2 are C(.dbd.NH)NH(OH), and R.sub.1 and R.sub.2 are bond.

30. The **composition** of claim 4, wherein L.sup.1 is

--CH.sub.2O--, W.sub.1 and W.sub.2 are C(.dbd.O)CF.sub.3, and R.sub.1 and R.sub.2 are bond.

31. The **composition** of claim 4, wherein L.sup.1 is --CH.sub.2CH.sub.2--, R.sub.1 and W.sub.1 together forms --(CH.sub.2).sub.aCH(NHR.sup.29)CO.sub.2H, wherein a is an integer from 0. . .

32. The **composition** of claim 31, wherein R.sub.2 and W.sub.2 together forms --(CH.sub.2).sub.bCH(NHR.sup.30)CO.sub.2H, wherein b is an integer from 0 to 2 and. . .

33. The **composition** of claim 32, wherein a and b are 1, and R.sup.29 and R.sup.30 are --C(.dbd.O)CH.sub.3.

34. The **composition** of claim 2, wherein said compound is of the formula: ##STR60##

35. The **composition** of claim 1, wherein said compound is of the formula: ##STR61##

36. The **composition** of claim 35, wherein said compound is of the formula: ##STR62##

37. The **composition** of claim 36, wherein Y.sup.1 is --NH.sub.2.

38. The **composition** of claim 37, wherein m and n are 0.

39. The **composition** of claim 1, wherein said compound is of the formula: ##STR63## wherein X.sup.1, X.sup.2, X.sup.3 and X.sup.4 are NR.sup.16.

40. The **composition** of claim 39, wherein said compound is of the formula: ##STR64##

41. The **composition** of claim 1, wherein said compound is of the formula: ##STR65##

42. The **composition** of claim 1, wherein said compound is of the formula: ##STR66##

43. The **composition** of claim 42, wherein R.sup.11 is lysine side chain residue, R.sup.12 is 2'-phenylethyl and R.sup.14 is --C(.dbd.O)CH.sub.3.

46. The method of claim 45, wherein said Fc **receptor** is selected from the group consisting of **Fc.gamma.** **RIIa**, FcγRIIb, FcγRIIc and mixtures thereof.

L3 ANSWER 11 OF 12 USPATFULL on STN
AN 2002:51012 USPATFULL
TI Fc receptor modulators and uses thereof
IN Baell, Jonathan B., Ivanhoe, AUSTRALIA
Garrett, Thomas P. J., Brunswick, AUSTRALIA
Hogarth, P. Mark, Williamstown, AUSTRALIA
Matthews, Barry R., Olinda, AUSTRALIA
McCarthy, Thomas D., East Malvern, AUSTRALIA
Pietersz, Geoffrey A., Greensborough, AUSTRALIA
PA Ilexus Pty Limited, Victoria, AUSTRALIA (non-U.S. corporation)
PI US 6355683 B1 20020312
AI US 1999-393598 19990910 (9)
PRAI US 1998-99855P 19980911 (60)
US 1999-131938P 19990430 (60)
US 1999-148479P 19990811 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Spivack, Phyllis G.
LREP Sheridan Ross P.C.

CLMN Number of Claims: 50
ECL Exemplary Claim: 1
DRWN 20 Drawing Figure(s); 20 Drawing Page(s)
LN.CNT 1876
CLM What is claimed is:

1. A pharmaceutical **composition** comprising: (a) compound having the formula: ##STR50## or salts thereof, wherein each of W.sup.1 and W.sup.2 is independently CO.sub.2R.sup.3 having. . .

2. The **composition** of claim 1, wherein said compound is of the formula: ##STR51##

3. The **composition** of claim 2, wherein said compound is of the formula: ##STR52##

4. The **composition** of claim 3, wherein W.sup.1 and W.sup.2 are CO.sub.2H.

5. The **composition** of claim 4, wherein R.sup.1 and R.sup.2 are a bond.

6. The **composition** of claim 5, wherein L.sup.1 is --CH.sub.2CH.sub.2--.

7. The **composition** of claim 5, wherein L.sup.1 is --CH.dbd.CHC(.dbd.O)--.

8. The **composition** of claim 5, wherein L.sup.1 is --CH.sub.2CH.sub.2CH(OH)--.

9. The **composition** of claim 5, wherein L.sup.1 is --CH.dbd.CH--.

10. The **composition** of claim 5, wherein L.sup.1 is --CH(OH)CH(OH)--.

11. The **composition** of claim 10, wherein the stereochemistry of hydroxy groups is (S,S).

12. The **composition** of claim 4, wherein R.sup.1 and R.sup.2 are --CH.sub.2--.

13. The **composition** of claim 12, wherein L.sup.1 is ethylene.

14. The **composition** of claim 12, wherein L.sup.1 is --CH.dbd.CH--.

15. The **composition** of claim 4, wherein R.sup.1 is methylene, R.sup.2 is a bond and L.sup.1 is ethylene.

16. The **composition** of claim 1, wherein the compound is of the formula: ##STR53##

17. The **composition** of claim 1, wherein the compound is of the formula: ##STR54##

18. The **composition** of claim 1, wherein the compound is of the formula: ##STR55##

19. The **composition** of claim 1, wherein the compound is of the formula: ##STR56##

20. The **composition** of claim 1, wherein the compound is of the formula: ##STR57##

21. The **composition** of claim 1, wherein the compound is of the formula: ##STR58##

22. The **composition** of claim 1, wherein the compound is of the

formula: ##STR59##

23. The **composition** of claim 1, wherein the compound is of the formula: ##STR60##

26. The method of claim 25, wherein said Fc **receptor** is selected from the group consisting of **Fc.gamma.** **RIIa**, FcγRIIb, FcγRIIc and mixtures thereof.

L3 ANSWER 12 OF 12 USPATFULL on STN
AN 1999:4639 USPATFULL
TI Method of inhibiting phagocytosis
IN Schreiber, Alan D., Philadelphia, PA, United States
Park, Jong-Gu, Drexel Hill, PA, United States
PA University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)
PI US 5858981 19990112
AI US 1996-657884 19960607 (8)
RLI Continuation-in-part of Ser. No. US 1995-483530, filed on 7 Jun 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-316425, filed on 30 Sep 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-129381, filed on 30 Sep 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: VanderVegt, F. Pierre
LREP Nixon & Vanderhye P.C.
CLMN Number of Claims: 29
ECL Exemplary Claim: 1,7
DRWN 21 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 1687
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
CLM What is claimed is:
. . . γ subunit of the IgE receptor FcεRI comprising introducing into lung cells of a mammal bearing said receptor a peptide **inhibitor** of Syk kinase, or a mimetic of said peptide, wherein said peptide comprises the sequence YXXL (SEQ ID NO:8), wherein. . . 2. The method according to claim 1 wherein said **inhibitor** is said peptide.

10. The method according to claim 1 wherein said **inhibitor** targets the interval region between the second SH2 domain and the catalytic (kinase) domain of Syk kinase.

22. The method according to claim 21 wherein said Fc **receptor** is FcγRI, **Fc.gamma.RIIA** or FcγRIIIA.

24. The method according to claim 23 wherein said Fc **receptor** is FcγRI, **Fc.gamma.RIIA** or FcγRIIIA.